64. (New) The method of claim 1, further comprising monitoring progression of Alzheimer's disease in the patient using MRI.

65. (New) The method of claim 35, further comprising administering an additional dosage of antibody responsive to a decrease in the measured level of antibody. .

66. (New) The method of claim 1, wherein the antibody is a human antibody of IgG1 isotype.

67. (New) The method of claim 58, wherein the antibody is administered with a carrier as a pharmaceutical composition to the patient.

## REMARKS

Claims 1-24, and 29-32 and 54-67 are currently under consideration. Claims 1, 30, and 32 have been amended, and new claims 56-67 have been added.

Support for new claims 56-67 is provided at, *e.g.*, the following locations in the specification: claim 56 is provided at *e.g.*, p. 29, line 1, and Example XI, p. 70; claim 57 is provided at *e.g.*, p. 14, lines 17-19 and 23; 58 is provided at *e.g.*, p. 14, line 20; claim 59 is provided at *e.g.*, p. 78, line 6-8; 60 is provided at *e.g.*, p. 28, lines 31 and p. 79, lines 31-32; claims 61-63 is provided at e.g., p. 79, line 26-34; claim 63 is also provided at *e.g.*, p. 14, lines 3-12, Example XI, p. 70; claim 64 is provided at e.g., p. 79, line 33, claim 65 is provided at e.g., p. 35, lines 22-34, claim 66 is provided at *e.g.*, p. 21, line 18; and, claim 67 is provided at *e.g.*, p. 31, line 18. Thus, the new claims contain no new matter.

The paragraph numbering of the office action is used in responding to the Examiner's remarks.

2. As requested by the Examiner a supplemental information disclosure statement was filed August 20, 2001 (Paper 28). Paper 28 corrects incomplete citations, and supplies references which appear to have been separated from the file.

3. Traverse of the election of species requirement is maintained insofar as it designates antibodies to Aβ and antibodies to epitopes within Aβ as being separate species. Antibodies to Aβ and antibodies to epitopes within Aβ are not mutually exclusive species as

required by MPEP 806.04(f). Antibodies to Aβ and antibodies to an epitope within Aβ (e.g., 1-10) are related as genus and species, not as mutually exclusive species. In other words, antibodies to Aβ1-10 (the species) constitute a subset of antibodies binding to Aβ (the genus). Although the Examiner provides some explanation why antibodies to Aβ may be patentably distinct from antibodies to a particular epitope within Aβ, and why a search for antibodies to Aβ would not necessarily be co-exclusive with antibodies to an epitope within Aβ, the Examiner has not addressed the fact that antibodies to Aβ and antibodies to an epitope within Aβ are related as genus and species rather than as mutually exclusive species. Accordingly, it is requested that the election of species be withdrawn between antibodies to Aβ and antibodies to epitopes within Aβ.

In addition, it is submitted that claims 35-37 have erroneously been withdrawn from consideration. Claims 35-37 were included in Group I (which Applicant elected) in the Restriction and Election of Species Requirement of September 29, 2000. Moreover, in this same paper, the Examiner indicated that claims 35-37 were generic to each of the designated species for which Applicant was required to elect (see p. 4, second paragraph). Accordingly, it is undisputed that claims 35-37 are within the elected Group I and are generic to the elected species of antibodies to Aβ. Therefore, no basis has been given or is apparent why claims 35-37 have been withdrawn from consideration, and it is submitted that they should be restored.

5-6. The Examiner states that reference to "to the patient" in claim 1 is unclear. This phrase has been moved to an earlier point in the claim for greater clarity.

Claim 30: The Examiner has correctly understood Applicant's intent and the claim has been amended for improved clarity.

Claim 32: The claim has been amended for improved clarity as suggested by the Examiner.

7-8. Claims 1-21, 24, and 29-32 stand rejected as allegedly anticipated by Nettleship. The Examiner states that Nettleship discloses antibodies to beta-amyloid peptides, particularly in

beta-sheet conformation, but also in random coil or alpha-helix formation, and pharmaceutical compositions for administration of the same. The Examiner asserts that the reference appears to be enabling for determination of suitable doses and routes of administration. The Examiner asserts that the patient population of Nettleship would inherently include humans of various risk factors, symptoms and ages as recited in claims 2-8. The Examiner also refers to claims 29-31 but the sentence is incomplete (office action p. 5, first paragraph, last sentence). This rejection is respectfully traversed.

Nettleship discusses several assays to identify compounds that inhibit the neurotoxicity of Aβ cells in culture (columns 4 and 5), and speculates that compounds thereby identified might be useful for treating Alzheimer's disease (col. 1, lines 49-51). The reference indicates that Aβ in beta sheet form is toxic in such assays, whereas Aβ in random coil form is only minimally so. The reference further speculates that two classes of antibodies might be useful in therapeutic and/or diagnostic methods. One class is antibodies that specifically bind to Aβ in beta sheet form without binding to Aβ in random coil form (col. 5, lines 45-47). The other class has the opposite binding specificity; that is, antibodies that specifically bind Aβ in random coil form without binding to Aβ in beta sheet form (col. 5, lines 47-50). The reference also speculates that the former class might be incorporated in pharmaceutical compositions (see abstract and column 2, lines 5-10). The reference is silent as to whether the second class of antibodies is similarly intended to be incorporated into pharmaceutical compositions. However, the fact that this class of antibody is specific for a form of Aβ that showed only "minimal" toxicity in the screening assays (see col. 5, lines 30-33) indicates that this class was not intended for therapeutic use, but at most for diagnostic use in combination with antibodies specific for the beta sheet form of Aβ.

To constitute an anticipatory reference, a prior art reference must place the inventors and thereby the public in possession of the invention (*In re Donohue*, 226 USPQ 619, 621 (Fed. Cir. 1985); *In re Sheppart*, 144 USPQ 42, 45 (CCPA 1964)). Possession of an invention involving a chemical genus "requires a precise definition, such as by structure,

formula, [or] chemical name of the claimed subject matter sufficient to distinguish it from other materials." *Regents of Univ. of Cal. v. Eli Lilly & Co.*, 43 USPQ2d 1398, 1405 (Fed. Cir. 1997). An "indication of a result that one might achieve if one made that invention" is insufficient to show possession of the invention. *Id.* at 1406.

Here, if the inventors of the Nettleship application were to attempt to pursue claims to a method of treating Alzheimer's disease, the claims ought to be rejected for lack of possession of what was being claimed. The application is directed to screening methods that might identify a class of compounds that might be useful for treating Alzheimer's disease. However, the class of compounds is identified only by desired functional properties of binding to Aβ in beta sheet form and not in random coil form, and presumably showing some activity in reducing toxicity of Aβ in beta sheet form in the assay discussed by the Nettleship application. No actual examples of suitable compounds are described. The description of compounds that might result from the assay and the hypothesis that such compounds might be useful in treating Alzheimer's disease are indications of the results one might desire to achieve if one were to make an invention. As is apparent from the *Lilly* case, description of a result that one might desire to achieve if one made an invention does not suffice to show possession of the invention.

In addition, a prior art reference cannot anticipate unless it provides an enabling disclosure of the claims at issue (*Chester v. Miller*, 15 USPQ 2d 133 (Fed. Cir. 1990)). Enablement is determined by balancing various factual determinations that may include (1) the quantity of experimentation necessary; (2) the amount of direction and guidance presented by the specification; (3) the presence or absence of working examples; (4) the nature of the invention (5) the state of the prior art; (6) the skill of those in the art; (7) the predictability of the art; and (8) the breadth of the claims. (*In re Wands*, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988) and *Ex Parte Forman*, 230 USPQ 546, 547 (BPAI 1986)).

Here, it is respectfully submitted that Nettleship does not provide an enabling disclosure of a method of treating Alzheimer's disease due to extent of experimentation required, the nature of the invention, the state of the art, the lack of direction and guidance provided by the

specification of Nettleship and the absence of working examples. To reiterate, Nettleship provides little guidance other than to indicate that one might administer a class of reagents having certain desired properties that one might obtain by certain screening methods to a patient as a means of treating Alzheimer's disease. Nettleship does not provide any working examples to illustrate his method, or even of compounds suitable for use in the method. Nettleship also does not address the issue of site of administration; in particular, whether compounds administered peripherally can be effective notwithstanding the obstacle imposed by the blood-brain barrier. The potential difficulties imposed by the blood brain barrier are evident from comments in the other references cited by the Examiner. For example, Friedland et al. states that they "do not anticipate passage of the labeled MAb through the blood-brain barrier" (p. 112, second column, first paragraph), and Walker indicates that the "development of techniques that facilitate the transport of large molecules across the blood-brain barrier is a potentially important step in refining strategies to allow access of ligands for Aβ to the brain from the blood" (at p. 382, first full paragraph).

Nettleship also does not indicate what end points might be used as an indication of successful treatment in a patient (for example, prevention of further deposition of Aβ in the brain, reduction of Aβ in the brain, reduction of toxicity to neuronal cells, or improvement in behavioral symptoms). In view of this lack of guidance, the practitioner is left to experiment with numerous variables simultaneously (agent, dosage, frequency, site of administration and end point). Although in some circumstances, the Examiner may be correct that it would be a routine matter to determine dosage and frequency of administration of a pharmaceutical, this is not the case when there are several other possible variables to explore. As the number of variables is increased, the number of permutations of variables increases exponentially. This results in a corresponding increase in the extent of experimentation required to find operable permutations. Moreover, this experimentation would have to have been performed in the context of a search for a first treatment of a hitherto untreatable disease for which considerable effort had already been expended in search of a cure. To have succeeded in devising actual compounds and a suitable regime for administration so as to prevent or treat Alzheimer's disease would have

been considered far from being a routine matter based on the limited discussion provided by Nettleship.

In addition, Applicant has attached a search report of the Derwent Inpadoc database showing the status of the Nettleship application in various countries. It can be seen that Nettleship has been abandoned in all jurisdictions. If the applicants of the Nettleship application did not feel possessed or enabled of a method of treating Alzheimer's disease, as might be inferred from its abandonment in all jurisdictions, then neither would a member of the public reading the application.

For the above reasons, it is submitted that Nettleship does not show possession or enablement of the invention as presently claimed, and therefore does not constitute an anticipating reference.

All dependent claims are distinguished for at least the same reasons as claim 1. Several dependent claims are further distinguished on independent grounds. For example, claim 5 is directed to administration to an asymptomatic patient. Nettleship proposes treatment only of patients already having Alzheimer's disease, and does not indicate that his proposed treatment would be effective prophylactically. Claims 29-30 specify administration of human antibodies prepared by immunizing a human with Aβ peptide. By contrast, Nettleship discusses mouse antibodies or antibodies derived therefrom such as humanized antibodies. Normally, one would not immunize a human with a human antigen due to the risk of generating an inappropriate immune response. It is the present application rather than Nettleship that indicates suprizingly that the immune response from immunizing a human with Aβ is in fact not inappropriate but rather advantageous in treatment or prophylaxis of Alzheimer's disease. Claim 56 specifies that the antibody is administered at a site separated by the blood-brain barrier from the brain. Although Nettleship refers to parenteral administration, he is not specific whether antibody is administered peripherally or directly to the brain. Both of these routes could constitute parenteral administration. The result that an antibody administered peripherally can be effective

in treatment of Alzheimer's disease is surprising in light of the limited amounts of antibody that cross the blood-brain barrier to reach the brain.

Claim 57 specifies that the antibody administered specifically binds to Aβ in dissociated form. By contrast, the antibody to be included in Nettleship's pharmaceutical composition binds to Aβ in beta sheet form but not to Aβ in random coil form. It is acknowledged the Examiner has noted that Nettleship also refers to a class of antibodies having the reverse specificity (i.e., binding to Aβ in random coil form but not beta sheet form). However, Nettleship's result that Aβ in random coil form was only minimally toxic would have suggested that these antibodies would not have activity in Nettleship's screening assay. Although Nettleship indicates that antibodies having specificity for Aβ in beta sheet formation are to be included in pharmaceutical compositions (see Abstract and p. 2, lines 5-10) he does not say the same for antibodies having the opposite specificity. In these circumstances, it appears that Nettleship's proposed class of antibodies having specificity for Aβ in random coil formation but not beta sheet formation are intended for diagnostic rather than therapeutic purposes. Certainly Nettleship does not manifest a clear intent that this class of antibodies is necessarily to be used for therapeutic purposes. Inherent anticipation cannot be found unless the "prior art *necessarily* functions in accordance with limitations of a process or method claim." (*In re King*, 23 USPQ 136, 138 (Fed. Cir. 1986)). (Emphasis supplied.)

Claim 59 specifies that administering of antibody reduced levels of Aβ in the brain of a patient. By contrast, although Nettleship refers generally to treating Alzheimer's disease, he does not disclose specific endpoints that might be observed. Due to the many uncertainties not addressed by Nettleship (e.g., which antibody to use, peripheral or intracranial administration, dosage, frequency) Nettleship does not necessarily achieve any particular outcome. Accordingly, Nettleship does not inherently anticipate claim 59 for this additional reason.

Claims 60-63 specify that administering of antibody results in an improved score by a pyschometric measure. As noted above, Nettleship discusses only a general goal of treating

Alzheimer's disease and does not disclose specific endpoints that might be achieved, or enable one necessarily to achieve a particular outcome.

Claim 61 specifies a human antibody having IgG1 isotype. Nettleship does not disclose human antibodies and particularly not the IgG1 isotype.

9. Claims 1, 9, 13, 20, and 22-23 stand rejected as allegedly anticipated by Friedland. Friedland is cited as teaching in vivo administration of murine monoclonal antibody 10H3 at a dosage of 10 microgram per mouse. This rejection is respectfully traversed.

"Anticipation is established only when a single prior art reference discloses, expressly or under principles of inherency, each and every element of a claimed invention," *RCA Corp v. Applied Digital Data Sys. Inc.*, 2212 USPQ 385, 388 (Fed. Cir. 1984). Inherent anticipation cannot be found unless the "prior art *necessarily* functions in accordance with limitations of a process or method claim." (*In re King*, 23 USPQ 136, 138 (Fed. Cir. 1986)). (Emphasis supplied).

Friedland does not expressly disclose administration of an antibody to Aβ to a patient in a regime effective to prevent or treat Alzheimer's disease. Rather, Friedland proposes that antibody be administered for purposes of in vivo imaging. In Friedland's own experiments, antibody was administered to post-mortem brains. Therefore, such treatment did not inherently prevent or treat Alzheimer's disease in a patient. Friedland does also consider hypothetically administering antibody to image living patients. However, Friedland acknowledges that a "noninvasive diagnostic method awaits development" (p. 112, first column, fourth paragraph) and that success is uncertain (*if* successful targeting occurs, SPECT imaging *may* provide a noninvasive measure of Aβ) (p. 112, second column, first paragraph, emphasis supplied). Further, even if an antibody were administered in a regime that achieved imaging, the same regime would not necessarily prevent or treat Alzheimer's disease. In vivo imaging simply requires delivery of sufficient labelled antibody to generate a detectable image. It is not necessary that the antibody clear or prevent deposition of Aβ; indeed, if the antibody were

completely successful in this regard, there would be nothing to label, thereby defeating the purpose of obtaining an in vivo image. In view of the fact that Friedland never carried out his method on a living human, and that the requirements of a regime for imaging would not necessarily achieve prevention or treatment of Alzheimer's disease, Friedland does not inherently anticipate the present claims.

10. The application names only a single inventor.

11. Claims 1-24 and 29-32 stand rejected as allegedly obvious over Nettleship. The office action repeats the remarks made concerning Nettleship for purposes of anticipation to which applicants have responded above. However, the Examiner has not indicated a secondary reference or general knowledge in the art to be combined with Nettleship nor indicated what motivation would support such a combination with a reasonable expectation of success. Applicants await further clarification of this issue. However, in the interim, Applicant notes that before the priority date of the present application, there was no effective treatment for Alzheimer's disease and little hope that one would be developed in the foreseeable future. Thus any prophetic proposal for treatment of Alzheimer's disease would have been regarded with skepticism at least. The Nettleship reference provides no experimental data to counteract such skepticism. The application does not even identify any compounds showing activity in its proposed in vitro assay, much less provide data showing signs of activity in vivo. Therefore, it is submitted that one would not have had a reasonable expectation of success that modifying Nettleship's teaching would result in prevention or treatment of Alzheimer's disease.

12. Claims 4 and 22-23 stand rejected as allegedly obvious over Walker. Walker is cited as teaching in vivo labelling of cerebral amyloid in primates using a mouse antibody at a dosage of 25 mg/kg. The Examiner acknowledges that Walker does not specifically teach administering antibodies to humans. However, the Examiner says that Walker suggests that it would be desirable to administer the antibody to humans for purposes of in vivo imaging. This rejection is respectfully traversed.

As discussed in connection with Friedland, administration of an antibody for purposes of in vivo imaging does not necessarily result in prevention or treatment of Alzheimer's disease. Therefore, one would have to modify Walker's imaging process not only by performing it on humans, but by altering the regime in such a manner as to prevent or treat Alzheimer's disease. For example, it is likely that antibody would have to be administered over a longer period to treat or prevent Alzheimer's disease than would be required to produce detectable label for imaging. Walker does not provide guidance as to how the regime might be altered for prevention or treatment of disease rather than imaging. Further, the expectation of success from altering Walker's own regime must be viewed in the context of a disease for which no treatment was available despite considerable commercial and humanitarian grounds for developing a treatment. In view of the lack of guidance of Walker as to alternative regimes for treatment, and the fact that Alzheimer's disease was viewed as being difficult if not impossible to treat, it is submitted that Walker does not provide a reasonable expectation of success of preventing or treating Alzheimer's disease.

If the Examiner believes a telephone conference would expedite prosecution of this application, please telephone the undersigned at 650-326-2400.

Respectfully submitted,

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**VERSION WITH MARKINGS TO SHOW CHANGES MADE**

Please replace the existing Cross-References to Related Applications section and replace it with the following replacement section.

[This application is related to USSN 60/067,740, filed December 12, 1997, USSN 60/080,970, filed April 4, 1998 and, 09/201,430 filed November 30, 1998, each of which is incorporated by reference in its entirety for all purposes.]The present application is a continuation-in-part of 09/201,430 filed November 30, 1998, which a nonprovisional of USSN 60/080,970, filed April 7, 1998, and a nonprovisional of USSN 60/067,740, filed December 2, 1997, each of which is incorporated by reference in its entirety for all purposes.

Please amend the claims as follows.

1. (Amended) A method of preventing or treating a disease characterized by amyloid deposit in a patient, comprising administering to the patient[an effective dosage of] an antibody that specifically binds to the amyloid deposit or a component thereof, in a regime effective [to the patient]to prevent or treat the disease.

30. (Amended) The method of claim 29, wherein the human immunized with Aβ peptide is the patient.

32. (Amended) The method of claim 1, wherein the [agent]antibody is administered intraperitoneally, orally, subcutaneously, intramuscularly, topically or intravenously.

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Basic Patent (No,Kind,Date): CA 2115900 AA 940823 <No. of Patents: 004>

PATENT FAMILY:
CANADA (CA)
Patent (No,Kind,Date): CA 2115900 AA 940823
PHARMACEUTICAL SCREENS AND ANTIBODIES (English; French)
Patent Assignee: LILLY CO ELI (US); ATHENA NEUROSCIENCES INC (US)
Author (Inventor): BECKER GERALD W (US); BREMS DAVID N (US); CHANEY MICHAEL O (US); MAY PATRICK (US); RYDEL RUSSELL E (US); SIMMONS LINDA K (US); TOMASELLI KEVIN J (US)
Priority (No,Kind,Date): US 21609 A 930222
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EUROPEAN PATENT OFFICE (EP)
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Patent Assignee: LILLY CO ELI (US); ATHENA NEUROSCIENCES INC (US)
Author (Inventor): BECKER GERALD WAYNE (US); BREMS DAVID NETTLESHIP (US); CHANEY MICHAEL OWEN (US); MAY PATRICK CORNELIOUS (US); RYDEL RUSSEL EUGENE (US); SIMMONS LINDA KAREN (US); TOMASELLI KEVIN JAMES (US)
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CA Abstract No: * 121(17)195902T; 121(17)195902T
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PHARMACEUTICAL SCREENS AND ANTIBODIES. (English; French; German)
Patent Assignee: LILLY CO ELI (US); ATHENA NEUROSCIENCES INC (US)
Author (Inventor): BECKER GERALD WAYNE (US); BREMS DAVID NETTLESHIP (US); CHANEY MICHAEL OWEN (US); MAY PATRICK CORNELIOUS (US); RYDEL RUSSEL EUGENE (US); SIMMONS LINDA KAREN (US); TOMASELLI KEVIN JAMES (US)
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| EP 613007 | P | 930222 | EP AA | PRIORITY (PATENT APPLICATION) (PRIORITAET (PATENTANMELDUNG)) US 21609 A 930222 |
| EP 613007 | P | 940218 | EP AE | EP-APPLICATION (EUROPAEISCHE ANMELDUNG) EP 94301170 A 940218 |
| EP 613007 | P | 940831 | EP AK | DESIGNATED CONTRACTING STATES IN AN APPLICATION WITHOUT SEARCH REPORT (IN EINER ANMELDUNG OHNE RECHERCHENBERICHT BENANNTE VERTRAGSSTAATEN) AT BE CH DE DK ES FR GB GR IE IT LI LU NL PT SE |
| EP 613007 | P | 940831 | EP A2 | PUBLICATION OF APPLICATION WITHOUT SEARCH REPORT (VEROEFFENTLICHUNG DER ANMELDUNG OHNE RECHERCHENBERICHT) |
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JAPAN (JP)
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PHARMACEUTICAL CLEANING AND ANTIBODY (English)
Patent Assignee: LILLY CO ELI; ATENA NIYUUROSAIENSUIZU INC
Author (Inventor): JIERARUDO UEIN BETSUKAA; DEIBITSUDO NETORUSHITSUPU BURE; MAIKERU OOEN CHIRINII; PATORITSUKU KOONERIASU MEI; RATSUSERU YUUJIIN RAIDERU; RINDA KAREN SAIMONZU; KEBIN JIEIMUZU TOMASERI
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